COMPARISON OF THIOPENTONE SODIUM VERSUS PROPOFOL AS INDUCTION AGENT FOR MODIFIED ELECTROCONVULSIVE THERAPY

A STUDY OF 60 CASES

DISSERTATION SUBMITTED FOR THE DEGREE OF

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DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"COMPARISON OF THIOPENTONE SODIUM VERSUS PROPOFOL AS INDUCTION AGENT FOR MODIFIED ELECTROCONVULSIVE THERAPY"** is a bonafide record work done by **Dr. M. SELVI ANNIE GEETA** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X –Anaesthesiology.

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DECLARATION

I, **Dr.M. SELVI ANNIE GEETA** solemnly declare that the dissertation titled **"COMPARISON OF THIOPENTONE SODIUM VERSUS PROPOFOL AS INDUCTION AGENT FOR MODIFIED ELECTROCONVULSIVE THERAPY"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.D. degree Branch X (Anaesthesiology) to be held in March 2008.

Place: Madurai **Dr. M. SELVI ANNIE GEETA**

Date :

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CONTENTS

INTRODUCTION

Electro convulsive therapy (ECT) is a treatment that has generated considerable controversy since its introduction to psychiatric practice in 1938. However a series of well conducted studies since the 1970s have conclusively established the efficiency of ECT in the treatment of affective disorders especially depression and to a lesser extent in the treatment of schizophrenia. Modifications of ECT practice over the last four decades have considerably improved its safety and efficacy.

Ever since “Modified electroconvulsive therapy”is introduced in 1963, by the use of Intravenous anaesthetic agents, neuro muscular blockade and assisted or controlled ventilation with oxygen, the ANAESTHESIOLOGIST has a significant role to play in this modality of treatment in psychiatry.

The Anaesthetic requirements for ECT are

1. Amnesia
2. Airway Management
3. Prevention of bodily injuries
4. Attaining haemodynamic stability
5. Smooth and rapid emergence

Anaesthesiologists must be equipped with an in-depth understanding of the changes affecting cardio vascular system, respiratory system and central nervous system by the treatment process and the pharmacological measures to attenuate them.

As the treatment process of ‘Modified electroconvulsive therapy’ is associated with significant haemodynamic disturbances, the Anaesthesiologist should reacquaint with relevant aspects of electro convulsive therapy, understand the effects of anaesthetic agents on the effectiveness of electroconvulsive therapy with an aim to acquire technical skill in this segment of Anaesthetic practice.

Electro convulsive therapy under general anaesthesia is associated with transient but significant hypertension and tachycardia. Cardiovascular derangements are due to lability of arterial blood pressure resulting in systemic complications in the susceptible patients with myocardial infarction, congestive cardiac failure and cerebrovascular accident. Haemodynamic lability is due to parasympathetic and sympathetic stimulation and adrenomedullary catecholamine release.

In clinical practice many different strategies have been advocated for modification of cardiovascular changes like administering beta blockers, calcium channel blockers, Lignocaine and fentanyl.

Use of different induction agent is one such strategy employed to obtund the cardiovascular response to ECT.

An ideal Anaesthetic agent for ECT should provide a smooth rapid induction, a rapid recovery and attenuation of the physiological effects of seizure activity.

Many centres in India use Thiopentone sodium which is an ultra short acting barbiturate. Now many other drugs like methohexitone, midazolam, etomidate and propofol were used as alternative induction agents.

In my study, I have compared thiopentone sodium and propofol as induction agents for modified electroconvulsive therapy.

AIM OF THE STUDY

To compare the induction agents, **thiopentone sodium** and **propofol** with regard to

a) Haemodynamic changes

b) Seizure duration

c) Recovery from anesthesia

d) Clinical outcome

In patients undergoing modified electroconvulsive therapy (MECT) for treatment of depression or mania.

HISTORY

Ladislas Meduna , a Hungarian Neuropsychiatrist and pathologist, in 1934 successfully treated a catatonic man by pentylene tetrazol (Metrazol) which induced epileptic fits.

Convulsive therapy was induced by insulin (Sakee) and with the inhalant Hexafluorodiethyl ether.

Electrically induced seizures, introduced by Cerelitti and Bini (1938) are the only form of convulsive therapy currently employed worldover.

In 1940, Bennett described the use of Curare for modifying drug induced convulsions and not electroconvulsions. Curare was subsequently used in electroconvulsive therapy.

During this period Bellett in 1941, Kolb in 1946 and Altschute in 1947 with their associates reported cardiovascular complications after convulsions. Further advances in modification of convulsion during ECT came with the introduction of gallamine by Hughenard and Bone in 1949 and introduction of succinylcholine by Holmberg and Thesieff in 1951.

Reports on the use of these drugs in electroconvulsive therapy preceded papers on their use in General Anaesthesia .

In 1959, Friedman reported the use on intravenous methohexital for modification of seizure activity.

By 1960's the technique of using short acting intravenous barbiturates and depolarizing muscle relaxants became accepted as simple, safe regime in order to produce modified eletroconvulsive therapy.

The early electroconvulsive therapy treatments were "Unmodified" i.e., neither implemented by sedation, anaesthesia, neuromuscular blockade nor by supplementary oxygenation and ventilation.

Beginning in 1963, the treatment was 'MODIFIED' by the use of intravenous anaesthetic agents, neuromuscular blockade, and assisted or controlled ventilation with 100% Oxygen. This gradually became the practice in most of the countries.

INDICATIONS FOR ECT

The primary indications of ECT are

a) Where a need exists for rapid, definitive response on either medical or psychiatric grounds,

b) When the risk of other treatments outweighs the risks of ECT

c) When a history of poor drug response and / or good ECT response exists for previous episodes of illness

d) The diagnostic conditions where ECT has been shown to be useful are :

I. DEPRESSIVE DISORDERS

People with unipolar and bipolar depressions show a 70% response to ECT. People given ECT to treat depression show a faster response than with antidepressants alone. ECT is indicated in depressed people with a high suicidal risk, who are in stupor, drug non-responders or partial responders, and are noncompliant. Depressed people with biological disturbances or psychomotor retardation or psychotic symptoms show the best response to ECT. Continuation therapy with antidepressants / lithium is required to prevent relapses after treatment with ECT.

II - MANIA

ECT combined with antipsychotics results in faster recovery than with antipsychotics or lithium alone. More frequent treatments are possibly associated with faster recovery than less frequent treatments. People with mania with severe disturbances show good response to ECT.

III – SCHIZOPHRENIA

ECT combined with antipsychotics produces a faster recovery from relapses in the first 6 weeks of treatment than antipsychotics alone, though this advantage is not always maintained over the long term. ECT may be of benefit in some people with schizophrenia who show a limited response to antipsychotics. When ECT is given with antipsychotics, an adequate clinical response is usually seen within 12 treatments, though in some instances, up to 20 treatments may be necessary. Patients with depressive, catatonic symptoms show good response with ECT, while people with chronic illness characterized by prominent negative symptoms may respond only minimally or not at all.

IV - SCHIZOAFFECTIVE DISORDERS

Evidence from retrospective studies shows that ECT may be useful in the treatment of acute episodes.

V – OTHER FUNCTIONAL PSYCHOSES

Acute psychoses usually respond well to antipsychotic medication which is the treatment of choice, but ECT is also effective, if otherwise indicated.

VI – SECONDARY DEPRESSION

Depression secondary to obsessive – compulsive disorder, severe anxiety states and reactive depressions, particularly if significant biological disturbances, psychomotor retardation and/ or psychotic features are present, may be given ECT if required.

CONTRAINDICATIONS FOR ECT

ABSOLUTE:	Recent Myocardial Infarction
	Recent Cerebrovascular accident
	Intracranial mass lesion
	Phaeochromocytoma
RELATIVE:	Angina pectoris
	Congestive heart failure
	Severe pulmonary disease
	Severe osteoporosis
	Major bone fractures
	Glaucoma
	Retinal detachment
	Pregnancy
	Thrombophlebitis

METHOD OF ADMINISTRATION OF ELECTROCONVULSIVE THERAPY

ECT is administered by a ECT machine. The waveform, frequency and duration of the electrical stimuli from ECT machine can be adjusted through a wide range to produce the type of generalized seizure intended. Current instruments are brief pulse instruments that use lower dosage of electrical energy. It involves flow of electrical energy only during each pulse duration, which is brief and of square wave form (MMECTA – Multiple Monitored Ect Apparatus / machine). A two channel chart recorder for EEG and ECG allows monitoring of the presence and duration of seizure activity as well as cardiac activity. In bilateral ECT, an electrode is placed over each hemisphere whereas in unilateral ECT, both electrodes are placed over one hemisphere.

NUMBER OF TREATMENTS

Usually a treatment schedule of six to eight ECT sessions spread over a period of two weeks. One or two day interval is left between two sessions. In resistant cases, maintenance ECT is also carried out.

MECHANISM OF ACTION

Generalised electrically induced seizures of the central nervous system are the sine qua non for the therapeutic effects of electroconvulsive therapy.

The psychological mechanism responsible for the therapeutic effect remains largely unknown.

EXPLANATIONS OFFERED FOR THE THERAPEUTIC EFFECT

a) NEUROPHYSIOLOGICAL ALTERATIONS IN

- Permeability of Blood Brain Barrier
- Cerebral microcirculation
- Neurometabolic activity
- Brain electrical activity

b) NEUROENDOCRINOLOGICAL

- Acute neuroendocrine discharge of ACTH, prolactin and hypothalamic peptides.

c) NEUROCHEMICAL CHANGES IN

- Ion transport systems
- Brain neurotransmitter receptor systems
- Biogenic amines

d) BETA ADRENERGIC RECEPTOR STIMULATION MECHANISMS

e) ELECTROLYTE CHANGES

PHYSIOLOGICAL EFFECTS OF ELECTROCONVULSIVE THERAPY

The physiological effects of electroconvulsive therapy can be classified under the following headings.

1. CARDIOVASCULAR EFFECTS
2. CEREBROVASCULAR CHANGES
3. NEUROENDOCRINE RESPONSES
4. SEIZURE RESPONSE
5. ENDOCRINE RESPONSE
6. ENZYME CHANGES
7. MISCELLANEOUS EFFECTS

The diagram alongside illustrates the physiological events following administration of the electroshock.

The following table gives the physiological consequences of electroconvulsive therapy.

TABLE ILLUSTRATING THE PHYSIOLOGICAL CONSEQUENCES OF ELECTROCONVULSIVE THERAPY

CARDIOVASCULAR EFFECTS

Immediate: parasympathetic stimulation*

Bradycardia

Hypotension

Asystole (possible)

Late (after 1 minute): sympathetic stimulation**

Tachycardia

Hypertension

Arrhythmias

↑ Cardiac output

↑ Myocardial oxygen consumption

CEREBRAL EFFECTS

- ↑ Cerebral oxygen consumption (CMRO2)
- ↑ Cerebral blood flow (CBF)
- ↑ Intracranial pressure (ICP)

MISCELLANEOUS EFFECTS

↑ Intragastric pressure

↑ Intraocular pressure

* Occurs immediately after passage of current

** Occurs within minutes.

CARDIOVASCULAR EFFECTS

Cardiovascular changes may be the most important and dangerous complications of ECT. They are more common in elderly patients and patients with co-existing cardiovascular disease.

The typical cardiovascular response to ECT consists of generalized autonomic nervous system stimulation with an initial parasympathetic induced bradycardia lasting 10 to 15 seconds followed immediately by a more prominent sympathetic response that is associated with the release of catecholamines and occasionally cardiac arrhythmias.

Arrhythmias during the parasympathetic phase can include several seconds of asystole, bradycardia, premature ventricular contraction (PVC), or ventricular escape. Other sign of increased vagal tone (eg. Hypotension) may be present.

During the subsequent sympathetic stimulation and clonic muscle phase, the increase in circulating catecholamine is directly related to the current intensity.

The most frequent arrhythmias are sinus tachycardia, ventricular tachycardia and premature ventricular contractions (PVC). Sinus tachycardia can vary from 20 to 115 percent of the preshock heart rate; peaks approximately 2 minutes post shock, and is usually self-limited.

Systolic and diastolic blood pressures are increased by 30-40 percent. There is a two to four fold increase in the rate pressure product (RPP) an index of myocardial oxygen consumption. Arterial blood pressure, heart rate and arrhythmias decline in parallel with the fall in plasma catecholamine levels. Occasionally arrhythmias and hypertension can persist. Whole body and myocardial oxygen consumption rates increase during ECT and venous return is decreased during the convulsion. The clinical significance of these changes in oxygen consumption and cardiovascular function will depend on the patient's general condition. This rise in circulating catecholamine can be blunted by anaesthesia.

CEREBROVASCULAR CHANGES:

Cerebrovascular changes include a brief period of vasoconstriction when the electrical stimulus is applied, followed by a sustained increase in cerebral metabolism and blood flow. The increase in cerebral blood flow can result in an increase in intracranial volume and intracranial pressure which is of concern in patients with intracranial mass lesions, increased intracranial pressure from any cause or cerebrovascular anomalies.

NEUROENDOCRINE RESPONSES:

Electroconvulsive therapy activates noradrenergic systems, enhances dopamine receptor sensitivity and reduces serotonin uptake. ECT activates the peripheral autonomic nervous system and causes release of secretions from many endocrine glands.

NEUROENDOCRINE RESPONSES TO ECT

1. An immediate release of ACTH with peak plasma levels at 2 to 5 minutes, which returns to normal by 45 minutes.
2. An increase in PLASMA CORTISOL, level with peak at 30 minutes, returns to normal in 2 to 4 hours
3. An increase in PLASMA EPINEPHRINE concentration to 15 time baseline by 1 minute, which returns to normal at 10 minutes.
4. An increase in PLASMA NOREPINEPHRINE level to three time baseline at 1 minute, which returns to normal by 20 minutes.
5. Transient increased release of glucagons and inhibition of glucose-mediated insulin secretion.

SEIZURE RESPONSE

The waveform, frequency and duration of the electrical stimulus from the ECT machine can be adjusted through a wide range to produce the type of generalized seizure intended.

Preceded by a latent period of 2-3 seconds, a bilateral grandmal convulsion ensues, a tonic phase of 10-20 seconds followed by a clonic phase of 30-50 seconds.

Reducing the duration of convulsion activity would result in the reduction of therapeutic effect as shown by the various studies.

The threshold has been clinically lower in males than in females, and lower in younger than elderly patients.

ENDOCRINE RESPONSES:

The therapeutic efficacy of electroshock has been ascribed to a variety of endocrine changes occurring during therapy, most of which have no clinical side effects.

The notable exception is the effects of ECT on Diabetes mellitus. In the diabetic patient,electrically induced seizures produce elevations of a rapid order of circulating catecholamines and cortisol, a transient rise in glucagon levels, and inhibition of glucose-mediated insulin secretion.

All of these effects can result in a relative hyperglycaemia in the diabetic patient. Since ECT appears to cause a variable effect on glucose levels in diabetic patients, vigilance in the medical management of the diabetic patient during a series of ECT treatment sessions is necessary. Blood glucose levels of all diabetic patients requiring ECT should be

carefully monitored during the series and for as long as 3 weeks after termination of the treatment.

ENZYME CHANGES

Although cardiac complications have been frequently implicated in the morbidity and mortality, ECT per se does not appear to be involved in direct cardiac tissue damage.

Following ECT, The total CPK concentration frequently increases, with the highest values at 6 hours, and returns to normal values by 48 hours. However there is no elevation of the isoenzyme CPK-MB which indicates myocardial damage. There is no elevation of LDH 1 and LDH2 and serum glutamate transaminases.

MISCELLANEOUS EFFECTS:

Electroconvulsive therapy produces significant elevations in intraocular pressure subsequent to the onset of the seizure. Elevated intragastric pressure is also an inevitable accompanying effect of the convulsion.

COMPLICATIONS OF ECT

- Fear and anxiety at induction
- Muscle aches and headache
- Memory disturbances – Anterograde and Retrograde
- Damage to teeth, tongue, eyes, cutaneous structures
- Prolonged seizures
- Pulmonary aspiration
- Laryngospasm
- Prolonged apnoea

CARDIAC COMPLICATIONS

These form the main reason for mortality in electroconvulsive therapy. Cardiovascular mortality has been reported to be 0.03%. The complications include Atrial arrythmias, AV dissociation, ST segment depression and a variety of changes and these have been discussed elaborately in the section on physiological effects of ECT.

PRINCIPLES OF ANAESTHETIC MANAGEMENT

General anaesthesia for electroconvulsive therapy is intended to provide the patient with lack of awareness of the electrical treatment, modification of the motor effects of the seizure in order to prevent injury, rapid recovery and minimal side effects and compatibility with medications the patient is taking.

Brief general anaesthesia has added to the safety and comfort of modified electroconvulsive therapy. While a simple procedure, there are several areas in which ECT differs from other procedures requiring brief anaesthesia. These differences call for even more collaboration, interaction and cooperation between an anaesthesiologist and psychiatrist than is customary.

Though the technical aspects of anaesthesia for electroconvulsive therapy seem straight forward and are of relatively minor complexity in an anaesthesiologist's overall practice, there are four areas which should command particular attention from anaesthesiologists.

First, while only a short time period is involved in modified electroconvulsive therapy when compared to other medical procedures requiring brief anaesthesia.

Secondly, both the physiological and pathological aspects of the anaesthetic process itself may influence the actual treatment outcome.

Thirdly, because of the possible effect of anaesthesia on electroconvulsive therapy and the relative brevity of the treatment itself, more preliminary and on-the-spot coordination between anaesthesiologist and psychiatrist is necessary.

Fourth since the procedure is repetitive, an optimum anaesthetic regimen can be sought for each patient.

Anaesthetic requirements for successful modified electroconvulsive therapy are fourfold.

- Rapid and smooth induction
- Attenuation of the physiological effects of ECT
- Rapid recovery after the seizure
- Minimization of any antagonistic effects on seizure activity by anaesthetic agents.

ASSESSMENT OF PATIENTS FOR ANAESTHESIA BEFORE ECT

All patients subjected to ECT should undergo a thorough physical examination and a detailed medical history should be taken. Particular attention should be paid to cardiorespiratory function, allergies and previous anesthetic experience and a full neurological assessment should be made. The presence or absence of loose or missing teeth should be recorded. The patient should give written consent to the procedure.

Minimum Investigation that are done

Haemoglobin

- Urine
  - Alb
  - Sugar
- Blood
  - Urea
  - Sugar
  - Creatinine
- Chest X ray

Other tests should be performed if clinically indicated.

Records should be kept for each administration. This should include details of the drugs given and their effects, the nature and duration of the convulsion, the heart rate and blood pressure during the treatment and subsequent 60 minutes and any side effects or complications encountered.

PREANAESTHETIC PREPARATION

The immediate preanaesthetic preparation of the patients, as for any anaesthetic, must include a period of fasting of at least 6 hours. This may seem simple, but many of these patients are extremely unreliable and occasionally uncooperative. Careful supervision of the patient is required to ensure that fasting does occur. Most of the ECT sessions are scheduled in the early morning to minimize the fasting period.

Preanaesthetic medication with sedatives or narcotics is not required and may serve only to prolong the anaesthetic recovery time. Reassurance from the psychiatric staff should be sufficient to allay most of the patient’s fears regarding the upcoming treatment.

MONITORING AND CONDUCT OF ANAESTHESIA

Intravenous access is secured.

Monitoring includes pulse rate, arterial blood pressure, ECG and arterial oxygen saturation as a minimum. Full equipment to do Cardiopulmonary resuscitation (CPR) must be available. All the emergency drugs should be checked and kept ready.

The ideal intravenous anaesthetic agent would provide rapid onset short duration, attenuation of adverse physiological effects of ECT, rapid recovery and no adverse shortening of seizure duration. The barbiturates like thiopentone sodium and methohexital have been used. Propofol, as an alternative IV induction drug is also preferred.

The use of neuromuscular blocking drugs has attenuate muscle contractions associated with ECT and essentially eliminated the risks of fractures or other injuries associated with muscle contractions. Succinycholine in the dose of 0.5 – 1 mg/kg has been used most often. The dosage should be modified on an individual basis.

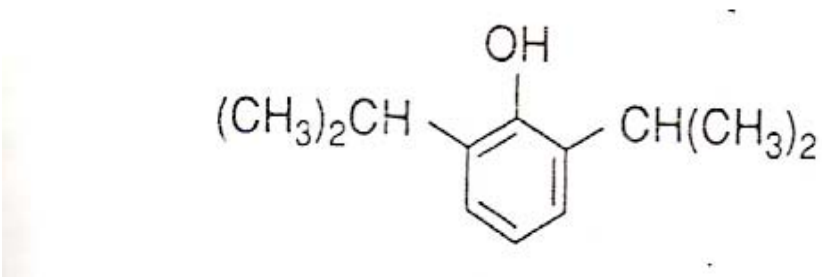
Ventilation with an oxygen enriched mixture should be approximately assisted or controlled upto the time of the seizure and again following recovery from the seizure.

It is important to monitor patients closely immediately following ECT and during the recovery room until they have recovered from anaesthesia.

PHARMACOLOGY OF PROPOFOL

Propofol is a substituted Isopropyl phenol (2-6 – Disopropyl Phenol)

It is an intravenous sedative and hypnotic



Commercial Preparation :

10% soyabean oil, 1.2% purified egg phosphate, 2.25% glycerol

Mechanism of Action :

Propofol is a selective modulator of GABA. GABA is an inhibitory neuro transmitter of the CNS. They increase transmembrane chloride conductance which result in hyperpolarisation of the postsynaptic cell membrane and functional inhibition of the post synaptic neuron.

Pharmocokinetics :

Metobolism of propofol is extremely rapid. After 30 minutes less than 20% of IV bolus dose of propofol remain in the circulation as the

uncharged compound. The drug is completely and rapidly metabolized to its sulphate and glucournide compounds and other related compounds that are eliminated via the kidney. It is also been studied that the metabolism of drug is not markedly deranged in patients with moderate degree of renal and hepatic dysfunction. Generally, clearance exceeds the capacity of liver blood supply suggesting that extrahepatic mechanisms constitute to the clearance of propofol from the blood.

Effect on Organ systems :

Cardiovascular system :

Propofol cause decrease in systemic blood pressure with corresponding decrease in cardiac output and systemic vascular resistance.

Negative ionotropic effect is due to decrease in intracellular calcium availability due to inhibition of sacrolemmal Ca++ shifts. Cardiac index remains unaltered.

Respiratory systems :

Propofol produces dose dependent depression of ventilation and apnea may occur. Propofol decreases the tidal volume and frequency of breathing. The ventilatory response to hypercarbia and arterial hypoxemia are decreased by propofol.

It produce bronchodilatation and decreases the incidence of intra operative wheezing. Hypoxic pulmonary vasconstriction is intact.

Hepatic and Renal function :

Propofol does not affect the hepatic or renal function. Prolonged infusion of propofol may result in excretion of green urine and uric acid excretion is increased.

Central Nervous System :

Propofol decreases the cerebral metabolic rate for oxygen (CMRO2), cerebral blood flow and ICP. It also decreases the systemic BP and hence decreases the cerebral perfusion pressure.

The effect of propofol on EEG activity is controversial. It has direct anticonvulsant effect that is dose dependent. Propofol also resulted in a shorter duration of motor and EEG activity after electro convulsive therapy.

Propofol also has neuroprotective effect – Propofol administered resulted in better burst suppression and better neurological outcome.

Clinical Uses:

Total Intravenous Anesthesia: Propofol has a short context sensitive half life and also has a short effect site equilibrium time thus making it a readily titratable drug for IV sedation.

The short context sensitive half life of propofol even with prolonged period of infusion combined with the short effect – site equilibrium time make this a readily titrable drug for IV sedation. Emergence from anaesthesia was rapid (time from end of infusion until patient being oriented was 12.8 – 18.9 min. In the recovery room patients were described as fully alert and oriented.

Side effects:

1. Allergic reaction

Allergic component of propofol due to the phenyl nucleus and Disopropyl side chain

2. Bacterial growth:

Propofol supports the growth of Escherichia coli & Pseudomonas aeuroginosa – aseptic precaution during handling is a must.

3. Antioxidant property

Presence of phenolic group inhibits lipid peroxidation and scavengers free radicals.

5. Pain on Injection:

Pain on injection is more when the drug is injected into a smaller vein. Using 1% lidocaine, short acting opioids and changing the composition of the carrier fat emulsion to long and medium chain triglycerides decrease the incidence of pain on injection.

PHARMACOLOGY OF THIOPENTONE SODIUM

Sodium 5 – ethyl – (1- methylbutyl) 2- thiobarbiturate

Invented in 1934 by Lundy and Waters

Derivatives of bartituric acid, formed by condensation of urea and malonic acid

Week acid – with pka above plasma pH making them largely unionised and hence able to cross the blood brain barrier.

Dose of thiopental:

The dose of thiopental varies between 3 and 5 mg/kg, with an effective plasma concentration of 15mcg/mL.

Pharmacodynamic effects:

Barbiturates are positive allosteric modulators at GABA and glycine receptors. They cause increased channel opening time for chloride, which increase inhibitory effects.

Pharmacokinetics:

The effect compartment equilibrium half-time for thiopentone is very short – 1.2 minutes, which is faster than for propofol.

Thiopentone is highly protein bound depending on pH. Binding falls as pH rise. Clearance is by hepatic metabolism, not renal excretion

(<1%). Plasma concentration falls rapidly after a bolus dose of thiopentone owing to uptake by vessel rich tissue.

Hepatic extraction ratio is low. It undergoes oxidation to the carboxylic acid derivative and to a lesser extent by S – Oxidation to Pentobarbital, a hypnotic oxybarbiturate will slow elimination.

After a single bolus dose or short infusion pharmacodynamic decay curves follow typical first order kinetics. With longer, high-dose infusion hepatic metabolic capacity may be exceeded and zero-order kinetics may be seen. Desulphuration may then become a significant metabolic pathway. Recovery will be prolonged as a result of both reduced metabolism and the presence of an active metabolite.

PHARMCODYNAMICS:

Central Nervous System:

Central effects of thiopentone include sedation, anaethesia, anticonvulsion action, retrograde amnesia and depression of the vasomotor centre.

It is a cerebral vasoconstrictor causing a reduction in cerebral blood flow and intra cranial pressure and depression of cerebral metabolism. There are features of neuro protection. Burst suppression of EEG can be induced with high doses.

Respiratory system :

Thiopentone causes centrally mediated respiratory depression and reduced sensitivity to raised CO2 which is dependent on dose and rate of injection. Transient apnoea is common. Laryngeal reflexes are intact. Coughing, laryngeal spasm and mild bronchoconstriction can occur particularly in asthmatics.

Cardiovascular system :

Myocardial depressed in a dose dependent manner. Peripheral vascular resistance falls, leading to reduced preload and cardiac output. There is hypotension and tachycardia, which is exaggerated if there is hypovolemia, tight aortic stenosis and tamponade.

Adverse Effects:

1. Respiratory depression, and airway obstruction. Laryngeal spasm is more common than with propofol. Oxygen is administered by gentle manual IPPV via a mask.

2. Circulatory collapse: This is usually due to a relative overdose causing vasodilatation and myocardial depression. It may also be due to anaphylaxis. Treatment: raise the legs; give oxygen by IPPV infuse fluids fast intravenously, administer inotropes.

3. Coughing: a sign of regurgitation, salivation or over-light anaesthesia. Hiccup occasionally seen.

4. True cutaneous allergy can occur either in the form of a scarlatiniform rash or as true angioneurotic oedema. Photosensitivity to thiopentone in patients recently exposed to sunlight has been reported.

5. Severe anaphylactic reactions (allergy).These reactions, although very rare, are dangerous. They may take the form of cutaneous manifestations, (rashes, weals, flushes, oedema), cardiovascular collapse (hypotension, tachycardia), bronchospasm, laryngospasm and muscle rigidity or abdominal pain.

6. Acute intermittent porphyria. Barbiturates may precipitate lower motor neuron paralysis and perhaps death in patients with porphyria and are absolutely contraindicated in them.

REVIEW OF LITERATURE

The first report on the cardiovascular complication ECT came from BELLETH in 1941, KELB in 1946 and ACTSHOTE in 1947.

BOEY WK, LAIFO, *Anaesthesia analog 1990, August, Department of Anaesthesia, University of Singapore.*

"Comparison of propofol and thiopentone sodium as anaesthetic agents for electroconvulsive therapy."

Propofol and thiopentone were compared as anaesthetic agents for electro convulsive therapy in 31 patients on four occasions in a repeated measure cross over study. The increase in systolic and diastolic arterial pressures and heart rate after treatment were significantly higher with thiopentone. Propofol gave a milder tonus and clonus during seizure when both treatments were considered together. The time to walk 10 meters at 20 minutes was significantly better with propofol.

BONADA et al, Journal *of ECT 19(3) 129-132, September 2003,*

Propofol seems to be a good intravenous induction agent of choice for ECT. Its pharmokinetic properties ensure a rapid and deep

anesthesia, of short duration, with a minimum side effects and a rapid recovery of good quality, suitable for short repetitive procedures.

Gracia, Edwin, Rodriguez, *Rev cub Med Mil Apr-June 2007.*

"Propofol versus Thiopentone in electroconvulsive therapy"

50 psychiatric patients compared thiopentone sodium and propofol in electroconvulsive therapy. Following variables were analysed, mean blood pressure, cardiac frequency and rhythm, duration of generalized seizure and recovery. There was a greater increase in these variables in the thiopentone group compared to the propofol group. Generalised seizure duration was 29.84 seconds in propofol group and 37.24 seconds in thiopentone group. Recovery times were 6.85 min for thiopntone and 8.16 minutes for propofol. Propofol was a better hypnotic for electroconvulsive therapy.

MITCHELL P, JORDA T, HICHIE I, BUITIE C, *Australian NZJ Psychiatry 1991 Jun 25(2)*

"Propofol as an anesthetic agent for ECT, effect on outcome and length of course"

The aim of the study was to investigate the effect of propofol on the response to ECT.

Records of 66 patients with primary depression treated with ECT, 37 of whom had been assessed prospectively with Pre and Post ECT Hamilton and Zung depression severity ratings. Despite demonstrating that the individual seizure duration was significantly reduced with propofol compared to thiopentone they found no evidence of reduced ECT efficiency with propofol.

Park HS, Lee JH, Lee KH, " *British Journal of Anaesthesia Oct 1999 (14(3:2), Department of Anaesthesia, Presbyterian Medical centre, Chonju, Korea.*

"Comparison of Propofol and thiopentone for electroconvulsive therapy - effects on haemodynamic changes and intraocular pressure."

20 patients were studied during courses of ECT administration, each patient receiving propofol or thiopentone. The induction dose was 1.6 mg / kg of propofol and 3mg/kg of thiopentone sodium. The induction dose, rhythm and intraocular pressure were checked before induction and after administration of succinylcholine immediately, 5

minutes and 10 minutes after ECT administration. Recovery time was also compared between these two groups.

Results: Mean arterial pressure was lower following propofol than thiopentone ($p<0.05$) immediately after ECT. Heart rate was lower following propofol than thipentone ($p<0.05$) immediately 5 minutes and 10 minutes after ECT. Intraocular pressure was lower following propofol than thiopentone ($p<0.05$) immediately 5 minutes and 10 minutes after ECT. Recovery time of propofol (6.5 ± 0.8 minutes) was shorter than thiopentone (7.5 ± 0.9minutes).

Conclusion: Propofol for ECT induction would seem to be an ideal drug, as it attenuates hypertensive responses and increases in intraocular pressure.

Kadoi. Y. Saito, Ide M. Sekimoto K. Sehi, *Anaesthesia Intensive care, 2003 April : 31 (2).* "Department of Intensive care medicine and Anaesthesia school of medicine, Gunma University, Japan.

"The comparative effect of propofol versus thipentone on left ventricular function during electro convulsive therapy".

The purpose of this study was to compare the effect of propofol versus thiopentone on haemodynamics during electroconvulsive therapy as estimated by echocardiography.

Twenty eight ASA 1 & 2 patients scheduled for ECT were randomly divided into two groups to receive propofol 1 mg / kg or thipentone 2 mg/kg. Cardiac functions were examined by transesophageal echocardiography prior to induction of anaesthesia and throughout ECT until ten minutes after the seizure.

Results:

In the thiopentone group, increased end-systolic area (ESA) and decreased fractional area change (FAC) were observed compared to the propofol group. They have concluded that lesser haemodynamic changes occurred after the propofol anaesthesia compared with the thiopentone anaesthesia during ECT.

Singeri Saito, Yuifi Kadoi, *Anaesthesia Analog 2000.* **"**The comparative effects of Propofol versus thiopentone on middle cerebral artery blood flow velocity during electroconvulsive therapy"

In this study, they continuously compared cerebral blood flow at the middle cerebral artery during ECT by using propofol versus thiopentone Anaesthesia.

In the study, seizure duration was shorter in the propofol group. The shorter duration with the propofol and hence a smaller energy demand, may be a cause for the minor increase in cerebral blood flow velocity in the propofol group. The systemic haemodynamic changes are small and hence they cause a minor change in the cerebral haemodynamics compared to thiopentone group.

Mitchell P. Smythe G. *Biol Psychiatry 1991 March 15,*

“Effect of Anaesthetic agent propofol on hormonal response to ECT”. Propofol is a new anaesthetic agent that reduces electroconvulsive therapy (ECT) seizure duration. They reviewed the endocrine response to ECT by two distinct mechanism, decreasing prolactin by reducing the seizure duration and decreasing ACTH and cortisal by another process, possibly via a reduction in central nor adrenergic activation.

FEAR CF LITTLE JOHNS CS, ROUSE E, MCQUAIL P. *British Journal of Psychiatry 1995 Marh 166(3) 399-401.*

"Propofol Anesthesia in electroconvulsive therapy"

"Reduced seizure duration may not be relevant"

In a prospective, randomized double blind study 20 subjects with major depressive illness received either propofol or methohexitone Anesthesia. The Hamilton depression rating scale was used to assess depression before therapy, at every third treatment and at the end of therapy. Seizure duration was measured using the cuff technique.

Results : Mean seizure duration and mean total seizure duration were shorter in the propofol group but there was no difference in the outcome.

Conclusion: Use of propofol may not adversely affect outcome from depression and it is not necessarily contraindicated as an induction agent for ECT.

JOURNAL PAKISTAN MEDICAL ASSOCIATION FEB 2005 ; 50 (2) 6:3

"Comparison of thiopentone sodium and propofol for electro convulsive therapy."

Study conducted in department of anaesthesiology and intensive care, Aga khan University Hospital, Karachi.

Twenty five patients each undergoing atleast 2 sessions of ECT at the psychiatry department were included in the study. Each patient either received thiopentone or propofol for induction of sleep in a randomized manner. Drugs were evaluated regarding their effects on ECT induced haemodynamic changes (Blood Pressure, heart rate, seizure duration related to the procedure and recovery from sleep. Any side effect during the procedure and recovery was also noted.

They have concluded that propofol offered superior haemodynamic stability during the procedure and a quick recovery from sleep and propofol was found to be a better induction agent for ECT compared to thiopentone sodium.

BUTTER FIELD NN, GRAFT P, Journal *of ECT, March 2004, 20(1),*

"Propofol reduces cognitive impairment after electro convulsive therapy"

Cognitive impairment is the main complication after electro convulsive therapy (ECT). Modification of treatment parameters has been shown to affect the magnitude of these impairments, but the role of Anesthetic type remains unclear. This study tested whether there is a difference in cognitive impairments immediately after ECT with propofol compared to thiopentone.

Method: This was a randomized double blind cross over study which included 15 patients receiving right unilateral ECT for depression. Patient received propofol or thiopental on alternating ECT up to 6 treatments. Immediate and delayed verbal memory, reaction speed, and executive functions were assessed 45 minutes after each ECT.

Result: Cognitive impairment was reduced after ECT with propofol compared to thiopentone. Time to emergence was quicker and EEG seizure duration shorter after propofol treatment. There was no significant correlation between seizure duration and neuropsychological test performance.

Gaines Gy, Rees DI, *Anaesthetic Analog 1986 ; 65: 1345-56*

"Electroconvulsive therapy and Anaesthetic consideration."

In this study, 85 patients who were undergoing ECT for major depression in ASA-I, II group were included in our study. They used thiopentone, etomidote and propofol in succession in each patient. The effect of these agents on motor and EEG seizure times, heart rate, mean BP and peripheral O2 saturation were compared. They have concluded that Propofol appears to be a safe anaesthetic for ECT with minimal side effects. It was found superior to thiopentone and etomidate in attenuating the physiological response to ECT with milder haemodynamic changes.

AlRezah, Alinjanpour E. *Journal of Babol University of Medical Science in (JBUMS) 2005, 7 (3(27).*

“Comparison of Recovery duration of Propofol and thiopentone in ECT.

The aim of the study was compare the recovery duration of Propofol and thiopentone sodium in ECT.

Mean recovery duration of propofol and thipentone sodium was 5.49±2.57 minutes and 6.4±3.69 minutes. They have concluded that propofol can prevent increasing haemodynamic response to ECT better than thiopentone sodium.

MATERIALS AND METHODS

Study Design:

Place & Period:

This study was done at the ECT treatment room in the Department of Psychiatry, Government Rajaji Hospital, Madurai attached to Madurai Medical College, Madurai from 2005 to 2007.

Sample Size:

Sixty patients

Inclusion Criteria :

Sixty patients (19-64 yrs) of ASA grade I and II patients diagnosed to have moderate to severe depression or mania in accordance with the international classification of disease 10^{th} revision (ICD-10) who was prescribed ECT by the treating Psychiatrist were included in the study.

Exclusion Criteria:

Patients with contraindication to ECT

Uncontrolled hypertension

Valvular heart diseases

Thyroid dysfunction

Allergy to sulfa drugs / egg protein

H/o Porphyria / Bronchial asthma

Those who were able to give written consent for ECT and anesthesia, written consent was obtained, and for those patients who were deemed unfit to give consent by the treating psychiatrist, consent was got from their relatives.

Sampling Procedure:

The sixty patients in this study were randomly allocated into two groups, one of which received thiopentone sodium and the other propofol as the anesthetic agent for ECT.

Randomisation was done using a stratified design. Patients were stratified according to whether they were diagnosed to have depression or mania. Randomisation was done by the Psychiatric PG who subsequently did not have anything to do with treatment allocation or outcome assessment.

Outcome assessment was done by trained raters who were blind to the drug used. Each patient's pre-treatment and post-treatment psychiatric ratings were done by the same rater.

The Institution's research and ethics committee approved the study. ECT was given thrice weekly on Tuesdays, Thursdays and Saturdays.

IMPLEMENTATION:

In all patients a detailed history, physical examination and relevant investigations were done and medication noted. Patients who were on benzodiazepines had the drug discontinued 12 hours prior to ECT. All patients were fasted overnight and received.

Resuscitative equipments and emergency drugs were kept ready before administering ECT to treat the complications if any that might occur.

1. Cylinders with full oxygen supply
2. Laryngoscope with appropriate sized blades
3. Endotracheal tubes of appropriate size and stylets
4. Airway and mouth prop of appropriate sizes
5. Functioning suction apparatus
6. Vasopressors and drugs for resuscitation

Intravenous access secured pre oxygenation was done. Pulse rate and oxygen saturation was monitored continuously using a pulse oximeter. Preoperative B.P, HR and SPO2 were monitored and recorded.

After preoxygenation anesthesia was induced with either of the two drugs. Intravenous atropine sulfate 0.6mg was given.

a) **Group A -** Received propofol **1.5** mg / kg body weight with 1 ml of 2% Lignocaine hydrochloride as the anesthetic agent. Propofol was used for each subsequent ECT in these patients

b) **Group – B:** Patients received **Inj. Thiopentone sodium** 2mg/kg body weight and continued to receive thiopentone sodium for each subsequent ECT.

A blood pressure cuff was applied to the right upper arm and inflated to 40 mm of Hg above systolic B.P prior to the injection of succinylcholine to isolate the limb for monitoring motor seizure.

In both the groups muscle relaxation was achieved with intravenous administration of 0.5 mg/kg / bd wt of succinycholine.

Heart rate was monitored by manually palpating the radial artery pulsation and blood pressure was measured using blood pressure monitor at regular time intervals as indicated.

A screen was put to separate the anaesthetist giving the drug and the anaesthetist who is performing the procedure and taking the reading.

PROCEDURE:

After establishing an I.V. line on the left forearm the calculated dose of propofol or thiopentone was given over a period of 20 seconds. The induction dose was considered adequate if the eyelash reflex was lost

after 30 seconds; otherwise additional agents were injected (with increments of 0.2 mg / kg body weight of propofol or 0.5 mg / kg body weight of thiopentone sodium)

The sleep dose was recorded for subsequent treatment. Suxamethonium 0.5 mg/kg was given after the cuffed forearm was isolated.

Patients were ventilated normally at the rate of 8-10 breaths / min with 100% O2. Once the fasciculation due to suxamethonium subsided a soft mouth prop was inserted, bitemporal electrodes were placed for ECT and bilateral ECT was administered using brief pulse bidirectional constant current stimuli above seizure threshold (sine wave type) was used to administer electric shock.

The duration of motor seizure was recorded by a stop watch as well as stimulus intensity and the number of re-stimulation required to achieve a motor seizure of atleast 15 seconds. Any patient who did not develop a bilateral tonic clonic motor seizure of atleast 15 seconds were restimulated with higher stimulus doses by increasing the duration of pulses until on adequate seizure was achieved and maximum of 3 restimulations were permitted at each session.

Oxygenation was performed between re-stimulations. Once the motor seizure subsided patient's ventilation was assisted with a facemask with 100% oxygen until the patient resumed spontaneous respiration.

Any side effects like pain on injection, abnormal movement, and prolonged seizure defined as seizure duration > 120 sec, vomiting, bronchospasm or laryngospasm was noted.

During recovery, after 30 minutes the patient was asked some simple questions which they were able to understand and comprehend to assess the patient's orientation and ability to talk. Also the patient's ability to walk to short distance unaided was assessed after 1 hour and graded by the recovery room nurse who remained blinded to the anesthetic agent given. The presence of prolonged post-ictal restlessness or confusion was also noted.

OUTCOME ASSESSMENTS

Hamilton Depression Rating Scale (HDRS) was assessed pre and post treatment scores for depressed patients. Modified Mania Rating Scale (MMRS) was recorded pre and post treatment for mania patients by trained raters who remained blind to the anesthetic agent used.

Rating Scales :

a) Hamilton Depression Rating Scale (HDRS):

It is one of the best known depression rating scales which is used to rate the severity of symptoms. It is a validated, internationally used assessment tool. It takes into account not only information from the patient but also from all other sources. The original version of the scale comprised of 21 items. But this is reduced to 17 because the items of depersonalization / derealization, paranoia and obsessionality were found to be uncommon and diurnal variation was felt to be more related to the form of the illness. Higher scores indicate more severe depressive symptom. A rating of less than 8 is usually taken to indicate the absence of significant depressive symptoms.

b) Modified Manic Rating Scale (MMRS) :

This is a 28-item scale with ratings made on the basis of severity based on clinical interview and on information gained from relatives and nurses. It has satisfactory psychometric properties with good inter-rater reliability and test-retest reliability and is sensitive to change.

Ratings were made in the mornings only to minimize the effects of diurnal variation of symptoms. The first rating was done within 48 hours

of the first. ECT treatment and the second was done within 2 weeks of the past ECT treatment.

The haemodynamic data was compared and analysed by the students‘t’ test. A ‘p’ value of < 0.05 was considered statistically significant.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002) developed by Centers for Disease Control and Prevention (CDC), Atlanta for W.H.O.

Using this software, frequencies, percentage, range, mean, standard deviation, x^2 and 'p' values were calculated. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATION & RESULTS

Sixty patients who underwent electroconvulsive therapy using propofol or thiopentone sodium as induction agents. All of them were of either ASA class I or class II.

Table – 1

Psychiatric Diagnosis of Patients in the two groups (n=60)

ICD-10 Diagnosis	Propofol	Thiopentone	Total
Depression with psychosis	6	5	11
Depression without psychosis	7	6	13
Mania with psychosis	9	11	20
Mania without psychosis	8	8	16
Total	30	30	60

Total 24(40%) patient were diagnosed to be depressives and 36 (60%) were maniacs.

DEMOGRAPHIC DATA

Table - 2

Demographical Data (n=60)

Parameter	Propofol	Thiopentone	Range	P value
Age (year) Mean ± SD	37.3 ± 14.52	32.6 ± 9.37	18-62	0.295
Weight (kg) Mean ± SD	53.79 ± 11.17	52.11 ± 11.70	29-85	0.667
Male	16	23		
Female	14	7		

The mean age, weight and sex distribution of the included subjects are presented in Table 2

Subjects in each group did not differ with respect to these variables.

Patients in the two group did not differ with respect to proportions of patients who were previous drug non-responders. (Propofol 8/30, Thiopentone – 10/30)

Equal number of patients in each group was on concurrent medication, antidepressants or antipsychotics or both

The group did not significantly differ in proportion of patients with pre ECT physical risk factors such as hypertension (Propofol 4/30, Thiopentone 3/30)

Mean dose of Propofol given was 76.20 mg (SD $\pm$ 14.76 mg) Range (60-100 mg). The mean dose of thiopentone given was 120 mg (SD $\pm$ 28.30 range 100-200mg). The mean dose of succinylcholine given was 32.3 mg (SD $\pm$ 6.57 mg) (range 20-50mg).

HAEMODYNAMIC PARAMETERS

Changes in heart rate before and after ECT (n=60)

Table - 3

Parameters		Mean + SD		't' test	P value
		Thiopentone	Propofol		
Pre ECT		90.30±9.38	90.50±8.36	0.44	0.903 Not significant
Post ECT	1st min	95.88±8.80	94.28±9.5	0.50	0.676 Not significant
	2nd min	108.81±6.99	95.38±9.72	0.14	0.043 significant
	5th min	106.75±6.09	97.37±6.19	0.64	0.063 Not significant
	10th min	105.38±89.25	89.25±7.03	5.287	0.04 Significant

Heart Rate

The comparison of changes in the heart rate between the two groups is depicted in table 3.

There was an increase in heart rate from the second minute on wards in both groups. There was a maximum increase in heart rate by the 5^{th} minute in both groups, with the thiopentone group registering a higher heart rate than the propofol group. The maximum differences in heart rate between the two groups were observed in the 10^{th} minute. The mean heart rate in the propofol treated patients almost touched the pretreatment values, while the mean heart rate with the thiopentone group was still elevated.

The difference in mean heart rate between the pre ECT and peak heart rate following ECT was 16.08 beats / min for thiopentone whereas it is 6.87 beats / min for propofol.

In ECT there is an initial increase in parasympathetic stimulation between 0 to 2 minutes followed by sympathetic stimulation by 5 to 8 minutes which is seen in my observation of the heart rate.

Table - 4

Changes in Systolic BP before and after ECT (n=60)

Parameters		Systolic Pressure Mean ± SD		Mean differences	't' value	Significance
		Thiopentone	Propofol			
Pre ECT		127.63±11.97	125.33±10.68	0.55	0.5866	Not significant
Post ECT	1st min	139.77±13.07	128.77±14.85	2.220	0.0018	Significant
	2nd min	154.1±24.5	138.9±23.1	2.570	0.0077	Significant
	5th min	148.3±14.42	134.6±9.97	2.643	0.04	Significant
	10th min	130.40±7.58	126.43±9.15	1.988	0.06	Not significant

Changes in Systolic BP before and after ECT

The comparison of systolic blood pressure pre induction and at various time intervals following delivery of ECT stimulus is shown in table 4

Baseline differences in B.P. between the two groups were not significant.

There was an increase in systolic BP following the administration of ECT in both groups, but the increase in systolic blood pressure is much higher with the thiopentone group compared to the propofol group.

There was maximum increase in BP at 2^{nd} min in both the thiopentone and propofol group which is more with thiopentone compared to propofol and it is statistically significant.

There was an gradual increase in BP from the 2^{nd} minute to 5^{th} minute with a statistically significant increase in BP in the thiopentone group compared to the propofol group.

The systolic BP touched the baseline pressure at around the 10^{th} min in both groups.

Table - 5

Changes in Diastolic blood pressure before and after ECT (n=60)

Parameters		Diastolic BP Mean ± SD		't' value	P value
		Thiopentone	Propofol		
Pre ECT		81.07±7.68	84.67±6.77	1.34	0.1849 Not significant
Post ECT	1st min	98.1±11.45	90.27±9.3	2.03	0.0467 Not significant
	2nd min	92.43±7.63	83.47±6.75	3.36	0.0014 Significant
	5th min	86.47±6.74	81.4±6.81	2.01	0.0486 Not significant
	10th min	83.6±5.48	79.53±5.24	1.74	0.0866 Not significant

Changes in diastolic blood pressure before and after ECT

The comparison of diastolic blood pressure pre induction and at various time intervals following administration of ECT is show in the chart.

Baseline values did not significantly differ in both the groups.

There was an increase in diastolic BP following the administration of ECT in both the groups with the increase in diastolic BP higher with the thiopentone group compared to the propofol group.

Diastolic BP touched the maximum level around 1-2 minutes.There was statistically significant increase in diastolic BP in the thiopentone group compared to the propofol group.

The diastolic BP started to decrease and reached the base line value by 8-10 minutes.

ECT TREATMENT DETAILS

Table - 6

Parameters	Mean + SD		't' test	P value
	Thiopentone	Propofol		
Mean seizure duration (sec) Mean + SD	41.79 ± 11.73	29.59 ±8.97	12.20	0.004 Significant
Max. stimulus Intensity (MS) Mean ± SD	165 ± 55.59	276 ± 90.99	4.08	0.0001 Significant
No.of patients who required restimulation	5 (n=30)	9 (n=30)	0.8789	0.3831 Not significant
Total no.of ECT treatment	6.47 ± 1.57	7.37±1.52	1.57	0.1222 Not significant

Mean Seizure duration :

The mean seizure duration was more with thiopentone group compared to propofol group and it was statistically significant. Though propofol resulted in lesser mean seizure duration compared to the thiopentone group. Use of propofol as an induction agent does not adversely affect the outcome from the illness as seen by the outcome scores (MMRS & DMRS Score).

Maximum stimulus intensity : (MS)

The maximum stimulus intensity that is required to produce seizure is recorded. The observations showed that propofol group needed more stimulus intensity compared to the thiopentone group which was statistically significant.

Restimulation :

Number of patients who required restimulations were more with the propofol group compared with the thiopentone group. 9/30 in propofol group and 5/30 in thiopentone group, but it was not found to be statistically significant.

Total number of ECT treatments required

The total number of ECT treatments required to produce the desired effect is more with propofol group compared with the thiopentone group, but it was not statistically significant.

Thus, propofol use resulted in higher stimulus intensities being used to elicit adequate seizure, shorter seizure duration and greater number of treatments and more number of restimulations were required compared to the thiopentone group.

Table - 7

RECOVERY CHARACTERISTICS

		Thiopentone	Propofol
a)	Ability to talk after 30 min (Impaired)	16/30	6/30
b)	Ability to walk after 1 hour (Impaired)	13/30	4/30
c)	Post ECT complication		
	i) Restlessness	2	-
	ii) Post ictal confusion	4	-
	iii) Head ache	1	2
	iv) Nausea / vomiting / aspiration	1	1

Recovery characteristics :

This chart compares the recovery characteristics of the two groups. The propofol group had early recovery and was able to talk and walk better at 20 minutes compared to the thiopentone group patients.

In the propofol group 6/30 patient had the impaired ability to talk after 30 minutes compared to 16/30 patients in thiopentone group.

The ability to walk unaided at 1 hour was impaired is 4/30 in propofol group compared to 13/30 in thiopentone group.

Post ECT complications were more in thiopentone group compared to propofol group.

In thiopentone group,	Restlessness (2)	Post ictal confusion (4)
	Head ache (1)	Nausea / vomiting (1)
In propofol group,	Head ache (2)	Nausea (1)

Table – 8

Out come characteristics

Comparison of pre ECT and Post ECT Hamilton depression rating scale (HRDS) in depressive patients between two groups

Parameters	HDRS score Mean ± SD		't' test	P value
	Thiopentone	Propofol		
Pre ECT	16.41± 14.98	18.93±16.22	0.43	0.6652 Not significant
Post ECT	4.17±4.59	5.07±4.56	0.59	0.5902 Not significant

Comparing the two groups showed that there is no difference in pre treatment scores in both groups. Both groups were also similar in post treatment mean HDRS scores (statistically insignificant). Thus there was no difference in outcome between the two groups.

TABLE - 9

Comparison of Pre and Post ECT Mania Rating Scale (MMRS)

Score in Mania patients

Parameters	MMRS Score Mean ± SD		't' test	P value
	Thiopentone	Propofol		
Pre ECT	46.40 ±16.85	56.28±20.19	9.88	0.22 Insignificant
Post ECT	10.60±13.81	14.42±21.52	3.82	0.2694 Insignificant

Analysis of the two groups revealed no differences in pre treatment scores in both groups.

There was no statistically significant difference between the MMRS score between propofol and thiopentone following ECT treatment.

DISCUSSION

This is a prospective study which compares Propofol with Thiopentone on haemodynamic parameters, seizure duration, recovery, complication and outcome in a mixed group of 60 depressed and maniac patients.

Both the groups were matched by demographic and clinical variables. Out of these 24 patients (40%) were diagnosed to be depressive and 36 patients (60%) were maniac patients.

Pulse Rate :

Electroconvulsive therapy and the resultant seizures evokes an autonomic reaction generating a parasympathetic and then sympathetic activation sequence. The phase of sympathetic activation and a 15 fold increase in circulating catecholamines results in severe tachycardia

Accordingly the maximum pulse rate registered in our study in a single patient was 159/minute from his basal pulse rate of 72 beats / minute.

There was an increase in mean heart rate from the second minute onwards for both thiopentone and propofol group with a maximum heart rate around the 5^{th} minute.

Though initial tachycardia is due to the direct adrenergic outflow through sympathetic ganglia, the sustained response is due to the further release of epinephrine from adrenal medulla

The difference in mean heart rate between pre ECT heart rate and peak heart rate was 16.08 beats / min for thiopentone group, whereas it is 6.87 beats/min for propofol. The finding of my study correlate with the studies of Boey WK Lai FO, (Anaesthesia analog 1990, Aug 45) who observed a statistically significant increase in heart rate in the thiopentone group compared to the propofol group. Similar findings were observed by Park Hs et al, (BJA Oct 1999, 14:32) who observed that propofol had fewer effects on haemodynamic changes.

In our series, in which 2% of patients who were hypertensive, there was a very significant attenuation of pulse rate. Thus, propofol use has a very significant protective effect on the cardiovascular system as a whole.

Systolic and diastolic blood pressure

Both systolic and diastolic blood pressure is observed to increase from 1-2 minutes post ECT. At 2 minutes post ECT, the increase in systolic blood pressure was 40% in the thiopentone group compared to 9% in the propofol group.

In the 2nd, 5th & 10th min readings there was increase of BP more in the thiopentone group compared to the propofol group.

All these findings correlate well with the previous studies which showed a statistically significant difference in Blood pressure rise in thiopentone group compared to the Propofol group.

In 2% of patients who were hypertensives, systolic blood pressure reached basal levels at 2 minutes post ECT in the propofol group whereas they registered a 26% rise in the thiopentone group.

The observations made in systolic and diastolic BP in my study correlate well with the study of Kadoi Y Sait et al, (Anaesthesia Intensive care 2003 April 31(2), who observed decreased end systolic area (ESA) and decreased fractional area change (FAC) with the propofol group compared to the thiopentone group. Similar observation made by Boey WK, Lai Ko, Bonada et al, also confirmed the above findings with lesser increase in mean systolic and diastolic pressure in the propofol group compared to the thiopentone group.

Mean Seizure Duration:

There was statistically significant difference in mean seizure duration with the thiopentone group compared to the propofol group.

It is observed that propofol group needed more stimulus intensity compared to the thiopentone group which was statistically significant.

Though the total number of ECT treatments required to produce the desired effect is more with the propofol group compared with the thiopentone group, it was not statistically significant.

These findings of decrease in mean seizure duration in the propofol groups were comfirmed by the studies of Fear CF Little Johns CS, et al (BJA, 1995, March) who observed that reduced seizure duration in propofol group may not be relevant. Similar observation was also made by Mitchell P. Smythe G. who observed a decrease in ACTH, prolactin and cortisol level in the propofol group compared to the thiopentone group which is due to the decreased mean seizure duration.

Recovery characteristics:

The propofol group had early recovery and was able to talk at 30 minutes and walk better at 1 hour compared to the thiopentone group.

Post ECT complications were more with the thiopentone group compared to the propofol group.

The earlier recovery with the propofol group compared to the thiopentone group were consistent with the findings of Butter Field NN et al (Journal of ECT March 2004; 20 (1)) who observed that propofol

would reduce cognitive impairment after electro convulsive therapy and hence earlier recovery characteristics. Similar observations were made by Gracia et al (Rev Cub Med Mil Apr. June 2007) who observed a earlier recovery with propofol compared to the thiopentone group.

Outcome Characteristics:

There was no statistically significant difference in Pre and Post ECT HDRS scores for depressive patients and pre and post ECT MMRS scores for maniac patients.

SUMMARY

Modified electro convulsive therapy used to treat major affective disorders produces significant haemodynamic disturbances.

Increase in heart rate and blood pressure can produce serious impact on the cardiovascular system and these would be more in hypertensives and these have to be attenuated. Hypertension and tachycardia resulting from ECT may cause potentially serious adverse reactions such as myocardial infarction and stroke.

Sixty patients were randomized to receive either propofol (n=30) and thiopentone (n=30) in a prospective study to assess the haemodynamic changes, seizure characteristics, complications and psychological outcome. Out of 60 patients, 24 were (40%) depressive patients and 36(60%) were maniac patients in accordance with the ICD-10 diagnostic guidelines, who underwent modified electroconvulsive therapy. Patients continued the prescribed medication and the number of ECT treatments were decided by the treating clinician based on clinical improvement. Heart rate and blood pressure were monitored before and at varying periods up to 10 minutes after each ECT treatment. Induction and recovery times and post ECT complications were systematically observed. Clinical assessment of depression and mania were done by

trained raters who were blinded to the treatment assignment before the course of ECT and within two weeks of the last ECT treatment.

The groups did not differ significantly on demographic and clinical variables before treatment.

The base line mean heart rate was 90.30/minute in the thiopentone group and 90.50/minute in the propofol group.

In the 2nd minute after ECT the mean heart rate was 108.81 (an increase of 18 beats) in the thiopentone group compare to 95.38 (an increase of 5 beats) in the propofol group which was statistically significant. In the 5th minute, the mean heart rate was 106.75/minute in the thiopentone group compared to 97.37 / minute in the propofol group. By the 10th minutes, the mean heart rate continued to be higher in the thiopentone group (105.38/minute). while in the propofol group the heart rate touched the base line (89.25minute) which is also statistically significant.

On comparison of the systolic and diastolic BP the pre ECT mean systolic BP was 127.63 mmHg in the thiopentone group compared to 125.33mmHg in the propofol group. The increase in BP in 1st minute was 139.77mmHg the thiopentone group, compared to 128.77mmHg in the propofol group, which was statistically significant. The mean systolic BP

was highest in the second minute was 154.1mmHg in the thiopentone group whereas in the propofol group it was 138.9mmHg. which is statistically significant.

Mean systolic pressure in the 5^{th} minute was 148mmHg in the thiopentone group compared to 134 mmHg in the propofol group which is also statistically significant. By the 10^{th} minute, BP recorded was 130.4mmHg in the thiopentone group while it was only 126.4mmHg in the propofol group. Thus BP almost touched the base line in the propofol group.

Patients with propofol group required higher mean stimulus intensity to elicit adequate seizures, which was 165ms in the thiopentone group compared to 276 ms in the propofol group. Propofol group recorded a shorter mean duration of seizures 41.79seconds in the thiopentone group compared to 29.59seconds in the propofol group.

Propofol group also had more number of restimulation (5/30) in the thiopentone group compared to (9/30) in the propofol group.

Propofol group had early recovery characteristics compared to the thiopentone group. The ability to talk oriented at 30 minutes was 14 out of 30 patients in the thiopentone group compared to 24 out of 30 patients in the propofol group. Similarly the ability to walk unaided at 1 hour was

17/30 patients in the thiopentone group whereas it is 26/30 patients in the propofol group. Thus, propofol group had early recovery characteristics compared to the thiopentone group and the post ECT complications were less with the propofol group.

Outcome of the study by evaluating the MMRS and HDRS score were similar in both thiopentone and propofol group.

The results of this study indicate that propofol is a safe induction agent for modified ECT with significant advantages over thiopentone with regard to haemodynamic changes, speed of induction and recovery from anaesthesia with little untoward complications associated with its use. While overall outcome was similar in patients treated with propofol and thiopentone, use of the former was associated with longer courses of treatment, shorter seizures and greater stimulus dosing used to ensure adequate seizures.

These findings as well as the relatively higher cost of propofol indicates that the use of propofol as an induction agent for modified ECT should be reserved for those patients in whom it is required that elevations is heart rate and blood pressure after ECT are to be minimized.

Propofol would be ideal when early post operative recovery would be required as in day care surgeries with minimal post ECT complications.

CONCLUSION

Modified electroconvulsive therapy used to treat major affective disorders produces significant haemodynamic disturbances. Tachycardia and hypertension due to sympathetic stimulation resulting from ECT may cause potentially serious side effects especially in patients with hypertensive heart disease, ischaemic heart disease and in patients with compromised cardiovascular function.

Propofol when used as an induction agent was associated with a lower increase in pulse rate and blood pressure, which would be advantages in patients in whom tachycardia and hypertension would be detrimental.

Routine use of propofol for ECT is not encouraged because use of propofol requires higher mean stimulus intensity to elicit adequate seizures, (276ms) shorter mean duration of seizure,(29.59sec) more restimulations (9/30) and more number of treatment compared to the thiopentone group and hence propofol may be the drug of choice for induction in patients in whom sudden haemodynamic changes following ECT are detrimental.

Propofol use resulted in earlier recovery from the effects of anaesthesia than did the use of thiopentone. This occurred in spite of the significantly greater stimulus dose used in propofol treated patients. The use of propofol results in earlier recovery and less post ECT complications, which is of importance in the treatment of large numbers of outpatients.

HAMILTON DEPRESSION RATING SCALE (HDRS)

1. DEPRESSED MOOD – Sadness, hopelessness, worthlessness
   0) Absent
   1) These feeling states indicated only on questioning
   2) These feeling states spontaneously reported verbally
   3) Communicates feeling states non-verbally (i.e. through facial expression, posture, voice and tendency to weep)
   4) Patient reports VIRTUALLY ONLY these feeling states in his spontaneous verbal and non-verbal communication.
2. FEELING OF GUILT
   0) Absent
   1) Self reproach, feels he has let people down
   2) Ideas of guilt or rumination over past errors or sinful deeds
   3) Present illness is a punishment. Delusions of guilt
   4) Hears accusatory or denunciatory voices and / or experiences threatening visual hallucinations.
3. SUICIDE
   0) Absent
   1) Feels like life is not worth living
   2) Wishes he were dead or any thoughts of possible death to self
   3) Suicide ideas or gesture
   4) Attempts at suicide (any serious attempts rates 4)
4. INSOMNIA EARLY
   0) No difficulty
   1) Complains of occasional difficulty falling asleep (ie. More than ½ hour)
   2) Complains of nightly difficulty falling asleep

5. INSOMNIA MIDDLE
   0) No difficulty
   1) Patient complains of being restless and disturbed during the night
   2) waking during the night (any getting out of bed rates 2,except for the purpose of voiding)
6. INSOMNIA LATE
   0) No difficulty
   1) Waking in early hours of the morning but goes back to sleep
   2) Unable to fall asleep again if gets out of bed
7. WORK ANDACTIVITIES
   0) No difficulty
   1) Thoughts and feelings of incapacity, fatigue or weakness related to activities, work or bobbies
   2) Loss of interest in activity, hobbies or work – directly reported by patient or indirect in listlessness, indecision and cacillation (feels he has to push self to work or join activities)
   3) Decrease in actual time spent in activities or decrease in productivity (in hospital, rate 3 if patient does not spend atleast three hours a day in activities, hospital job or hobbies, exclusive of ward chores.)
   4) Stopped working because of present illness (in hospital, rate 4 if patient engages in no activities except ward chores, or if patient fails to perform ward chores unassisted.

8. RETARDATION - Slowness of thought and speech impaired ability to concentrate decreased motor activity
   0) Normal speech and thought
   1) Slight retardation at interview
   2) Obvious retardation at interview
   3) Interview difficult

4) Complete stupor

9. AGITATION
   0) None
   1) Fidgetiness
   2) Playing with hands, hair etc
   3) Moving about, can't sit still
   4) Hand wringing, nail biting, hair-pulling, biting of lips
10. ANXIETY PSYCHIC
   0) No difficulty
   1) Subjective tension and irritability
   2) Worrying about minor matters
   3) Apprehensive attitude apparent in face or speech
   4) Fears expressed without questioning
11. ANXIETY SOMATIC – Physiological concomitants of anxiety such as :
   Gastrointestinal (dry-mouth, wind,indigestion, diarrhea, cramps, belching)
   Cardiovascular (palpitation, headaches)
   Respiratory (hyperventilation, sighing)
   Urinary frequency
   Sweating
   0) Absent
   1) Mild
   2) Moderate
   3) Severe
   4) Incapacitating

12. SOMATIC SYMPTOMS, GASTRO INTESTINAL
   0) None
   1) Loss of appetite but eating without staff encouragement. Heavy feelings in abdomen
   2) Difficulty eating without staff urging. Requests or requires laxatives or medication for bowels or medication for G.I. symptoms.

13. SOMATIC SYMPTOMS, GENERAL
   0) None
   1) Heaviness in limbs,back or head, backaches, headaches, muscle aches, Loss of energy and fatigability
   2) Any clear-cut symptom rates 2

14. GENITAL SYMPTOMS – Loss of libido, menstrual disturbances
   0) Absent
   1) Mild
   2) Severe

15. HYPOCHONDRIASIS
   0) Not present
   1) Self-absorption (bodily)
   2) Pre occupation with health
   3) Frequent complaints, requests for help etc,
   4) Hypochondriacal delusions

16. LOSS OF WEIGHT – RATING BY HISTORY
   0) No weight loss
   1) Probable weight loss associated with present illness
   2) Definite weight loss (according to patient)

17. INSIGHT
   0) Acknowledges being depressed and ill
   1) Acknowledges illness but attributes cause to bad food, climate, overwork, virus, need for rest, etc.
   2) Denies being ill at all

MODIFIED MANIC STATE RATING SCALE (MMRS)

0	=	Absent
1	=	Slight flavor or suspicion of pathology by clinician
2	=	Clearly noticeable but mild or occurring not more than twice during interview
3	=	Clearly present and occurring 3-4 times during interview
4	=	Definitely present and frequent
5	=	Continuous and gross

1. Is depressed
2. Is talking excessively
3. Restlessness
4. Make threats
5. Has poor judgement
6. Is hallucinating
7. Looks happy and cheerful
8. Seeks out others
9. Is distractable
10. Has grandiose ideas
11. Has grandiose delusions
12. Is angry
13. Is suspicious
14. Has delusions of persecution
15. Is active
16. Is irritable
17. Jumps from one topic to another
18. Shows flight of ideas
19. Is careless about dress and grooming
20. Has diminished impulse control
21. Verbalizes feelings of well-being
22. Makes plans
23. Demands contact
24. Is sexually pre-occupied
25. Is emotionally labile
26. Is religious
27. Is disinhibited
28. Has disturbed sleep

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PROFORMA

Name	Age	Sex	Weight	Height	Hospital No.

ICD-10 Diagnosis :

Indication for ECT : (Tick Appropriate)

a) High Suicidal risk
b) Drug non-response
c) Intolerance to Drug
d) To hasten recovery
e) Response only to ECT in previous episodes
f) Others

Current Drug Therapy :

Fasting Period :

Pre-Medication: Inj. Atropine 0.6 mg – (YN)

Induction Drug Given: Thiopentone / Propofol (with Dosage)

Inj. Succinylcholine : Dose

Induction Time		
Stimulation Intensity		
Seizure Duration		
No.of Restimulation		

Para meter	Pre ECT		Post ECT									
			1^{st} min		2^{nd} min		5^{th} min		10^{th} min		30^{th} min	
HR												
Systole BP Diastole												

Other features: (v) Hicoughs / Branchospasms / Cough

Recovery Features

No.	Action	Time	Ability
1.	Opening of eyes		
2.	Ability to obey vocal commands		
3.	Sit up unaided		
4.	Ability to walk		Impaired / unimpaired
5.	Ability to talk		Impaired/ unimpaired

Post ECT Complications: (Tick Appropriately)

Restlessness / post Ictal confusion / Head ache / Nausea / vomiting / Aspiration

Patient Outcome:

	HDRS	YMRS
Pre ECT		
Post ECT		

MASTER CHART ABBREVIATIONS

HR	-	HEART RATE
SP	-	SYSTOLIC BLOOD PRESSURE
DP	-	DIASTOLIC BLOOD PRESSURE
HDRS	-	HAMILTON DEPRESSION RATING SCALE
MMRS	-	MODIFIED MANIA RATING SCALE
IMP	-	IMPAIRED
UNIMP	-	UNIMPAIRED
N	-	NO
Y	-	YES

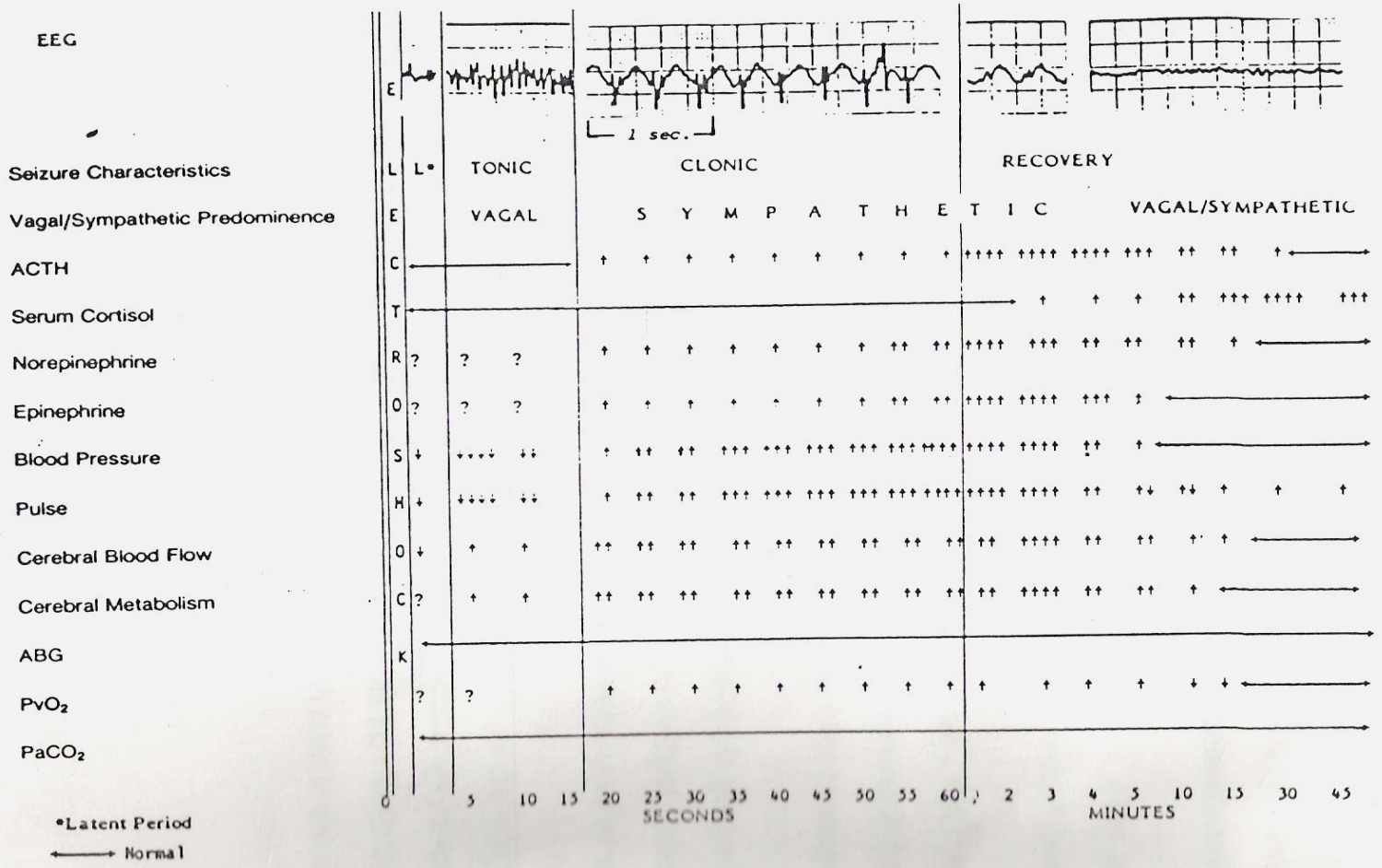

Fig. 70-15. A diagram of physiologic events following administration of the electroshock. (From Selvin,[121] with permission.)